

THE PREVALENCE OF NOISE INDUCED HEARING LOSS AND THE NEED FOR A
HEALTH PROMOTION/HEARING CONSERVATION PROGRAM FOR ADULTS

Capstone Document

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ABSTRACT

With the population of older adults over 65 years of age reaching 40 million individuals in 2010, much emphasis is being place on their health and well-being. A large number of this older adult population continues to work and/or still maintains an active lifestyle placing them at a higher risk of developing a noise-induced hearing loss. Noise-induced damage to the inner ear not only causes hearing loss, but also may create other auditory issues like tinnitus and hypersensitivity. Current research is making strides connecting the links between genetic factors and susceptibility, as well as pinpointing antioxidants and vitamins that may lessen susceptibility. Noise-induced hearing loss is a completely preventable condition with the use of adequate hearing protection. Because of this, there is a growing need for the development and implementation of a hearing health and conservation program for adults and older adults to become educated on and protect themselves against noise-induced hearing loss.

DEDICATION

This document is dedicated to my family and friends for their continual encouragement throughout graduate school. Without them I would not be leaving The Ohio State University with my sanity intact.

I would also like to give special thanks to my mother and sister for always telling me that I can do it in my moments of utmost procrastination.

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LIST OF ABBREVIATIONS
(Alphabetical)

ABR	auditory brainstem response
ALCAR	acetyl-L-carnitine
ANR	active noise reduction
ASHA	American Speech Language and Hearing Association
dB	decibels
IHC	inner hair cell
kHz	kilohertz
NAC	N-L-acetylcysteine
NHANES	National Health and Nutrition Examination Survey
NIDCD	National Institute on Deafness and Other Communication Disorders
NIHL	noise induced hearing loss
NIOSH	National Institute for Occupational Safety and Health
NRR	noise reduction rating
OHC	outer hair cell
OSHA	Occupational Safety and Health Administration
PTS	permanent threshold shift
ROS	reactive oxygen species
TTS	temporary threshold shift
WHO	World Health Organization

CHAPTER 1

Introduction

According to the United States Census Bureau, in 2010 the population of adults 65 years and older was over 40 million. It is projected that by the year 2020, this population will increase to 55 million and continue growth to 72 million in 2050. Of the 40 million elderly adults currently residing in the United States, a large number of them suffer one or more disabling chronic conditions (Administration on Aging, 2011). Currently, hearing loss falls within the top three most prevalent chronic conditions suffered in the older adult population, falling only behind hypertension and arthritis (Yueh & Shekelle, 2007). Depending on the method used to define a hearing impairment, 32% to 46% of the elderly population possess a measurable hearing loss (Cruickshanks et al., 1998). Fifteen percent of Americans between the ages of 20 and 69 suffer from a high frequency hearing loss related to harmful noise exposure through their occupation, recreational activities, or military services (National Institute on Deafness and Other Communication Disorders (NIDCD), 2008).

Noise-induced hearing loss (NIHL) is a completely preventable condition that occurs through permanent damage to important components of the cochlea (NIDCD,

2008). This underlying damage to the inner ear can lead to the development of speech understanding difficulties, tinnitus, and hypersensitivity (Mazurek, Olze, Haupt, & Szczepek, 2010; Burke & Creston, 1966). Researchers are beginning to gain more knowledge on NIHL and why some people tend to have a greater effect from noise exposure than others. Though some individuals may be at a higher risk of acquiring a NIHL due to their occupation or lifestyle, current research suggests that they may also be more susceptible to damage due to genetics, gender, or pigmentation (Konings, Van Laer, & Van Camp, 2009; Gallo & Gorig, 1964; Humes, 1984).

With the growing older adult population, many of them are at risk for NIHL through their occupations and leisure activities. Nondahl et al., (2006) assessed this topic in a population of older adults and found that older adults are more active than they once were. Because of this, they are now at a higher risk of acquiring a NIHL through noise-rich recreational activities. This study highlights the need for a hearing health program targeting the entire adult population with an emphasis on older adults. With proper education on adequate hearing loss prevention techniques and strategies, researchers in the future may witness the prevalence and incidence of NIHL slowly start to decline. The ideal goal of a hearing health program would be to educate individuals while they are young and continue educational programming throughout their lives. Though this hearing conservation program may be difficult to implement, gathering assistance from other medical personnel may help to reach a large majority of the adult population.

CHAPTER 2

Noise

Acoustically, noise is “complex sound waves with irregular vibrations and no defined pitch.” (American Speech-Language-Hearing Association (ASHA), 2011).

According to the Occupational Safety and Health Administration, noise is defined as an unwanted or undesired sound that could potentially harm ones body through excessive exposure (Occupational Safety and Health Administration (OSHA), 2011).

Using either definition, noise is a common pollutant that can be potentially damaging to one’s hearing without utilization of hearing protection (ASHA, 2011).

A publication on occupational noise exposure developed by the World Health Organization (WHO) highlights continuous and impulse noise as the two main types of exposure. Classification of noise exposure is dependent on two main characteristics: its’ duration over a specific period of time and whether or not the level of the noise remains constant or fluctuates. Continuous or constant noises are divided up into three subcategories: steady-state, fluctuating, and intermittent. Steady-state noise occurs when the noise is set at a fixed intensity level or has very minute fluctuations. When the fluctuations with the intensity level increase, the noise then becomes characterized as fluctuating. With intermittent noise exposure,

the signal includes a mixture of both steady-state and fluctuating noise (WHO, 2010).

Unlike continuous noise, an impulse noise exposure occurs over a short period of time and is defined as one or more short bursts of sound energy with a duration of one second or less. Harmful impulse noises are usually very loud with an intensity level of 120 dB or greater. Examples of an extreme impulse noise are gunfire or an explosion. Though exposure to continuous noise could potentially be damaging, unprotected exposure to high-level impulse noises usually results in instant and permanent damage to specific components auditory system like the tympanic membrane, ossicular chain, hair cells, basilar membrane, or stria vascularis (WHO, 2010).

CHAPTER 3

Anatomy & Physiology

Auditory stimuli travel a complex pathway from the outer and middle ear systems, through the cochlea, along the vestibulocochlear nerve, and ending in the primary auditory cortex located within the cerebral cortex of the brain. Although hearing for normally hearing individuals is effortless, it is actually an intricate and complex process. With damage to any part of the auditory pathway, an incoming sound stimulus may become distorted, unrecognized, or completely undetected (Henderson, Bielefeld, Harris, & Hu, 2006).

Sound travels throughout different parts of the auditory system where it is ultimately processed and distinguished by the brain. The auditory system can be broken down into two divisions, the peripheral auditory system and the central auditory system. The peripheral auditory system initiates at the pinna of the outer ear and concludes at the synapses between the auditory portion of the vestibulocochlear nerve and the neurons of the cochlear nucleus. The peripheral auditory system contains the outer, middle, and inner ear systems, which are equally important to the passage of sound. Depending on the location of the sound source and the individual's orientation in space, the pinna collects and funnels the

auditory stimulus into the outer ear where it then travels along the external auditory meatus until it reaches the tympanic membrane. The sound pressure level of the stimulus vibrates the tympanic membrane, which in turn activates a chain reaction in the middle ear system, vibrating the malleus, the incus and the stapes. (Musiek & Baran, 2007).

The middle ear system is formed around these three bones referred to as the ossicular chain. The ossicular chain transfers sound from the tympanic membrane into the oval window of the cochlea. The middle ear is an impedance transformer, matching the low impedance of air to the high impedance of the cochlear fluids.

The cochlea is the fluid-filled organ responsible for organizing and refining auditory stimuli (Musiek & Baran, 2007). Movement of the stapes in and out of the oval window creates movement of the cochlear fluids in the form of a traveling wave. The traveling wave reaches its maximum displacement at the location on the basilar membrane, which is “tuned” to the frequency of the stimulus. The outer hair cells (OHCs), tonotopically oriented along the basilar membrane, are responsible for amplifying the traveling wave and refining this area of maximum displacement for distinguishing the frequency of an auditory signal (Moore, 2007, ch.1). Though healthy OHCs are extremely important for the transduction process to occur properly, it is crucial that other structures of the cochlea, like the stria vascularis, remain intact. Not only does the stria vascularis help to supply the cochlea with an ongoing blood supply, it also maintains a chemical balance within the scala media through the production of endolymph. Without this balance, a breakdown in the

transduction process may occur (Henderson, Bielefeld, Harris, & Hu, 2006.) In a healthy cochlea, processing of auditory signals occurs without difficulty and the refined signal now travels along the auditory portion of the vestibulocochlear nerve until it reaches the brainstem. From there, the signal is directed to and further decoded through different structures within the brainstem and midbrain until it reaches the auditory cortex. Within the auditory cortex, the stimulus undergoes final frequency, temporal, and intensity decoding leaving the brain with the ability to distinguish and process the stimulus (Ades & Brookhart, 1950).

Damage to any area or structure in the auditory system, whether it is peripheral or central, can result in hearing loss, an auditory processing disorder, or other auditory conditions like tinnitus or hypersensitivity. A hearing loss as a result of excessive noise exposure is referred to as NIHL. This hearing loss is primarily classified as sensorineural, although a conductive component could be present in individuals who suffered an acoustic trauma from exposure to extreme levels of noise. In this specific population of individuals, the conductive component of their hearing loss is often a product of a perforated tympanic membrane or ossicular disarticulation (Cave, Cornish, & Chandler, 2007). A NIHL has detrimental effects to their inner ear system, specifically in the OHCs and inner hair cells (IHCs), stria vascularis, and basilar membrane (Henderson, Bielefeld, Harris, & Hu, 2006).

To fully understand NIHL, one must first understand the anatomical and physiological characteristics that result in this specific type of sensorineural hearing loss. Current research suggests that there are a number of events that occur within

the cochlea as a byproduct of excessive noise exposure. The events can occur at a cellular level with the damage and destruction of substantial numbers of OHCs and IHCs, as well as their supporting cells. Excessive noise exposure can also lead to a breakdown of the entire cochlea through the reduction or complete loss of blood flow, disruption in the chemical balance and, in more severe cases, the separation of the basilar membrane from the modiolus (Talaska & Schacht, 2007).

A normally functioning cochlea is responsible for receiving auditory input from the middle ear system, refining this input, and transmitting it along to the vestibulocochlear nerve. The structures that carry out this intricate and extremely vital process are the IHCs and the OHCs. The IHCs are responsible for transforming the mechanical stimulus from the defined movement of the basilar membrane into an electrical signal. This electrical signal is then sent along the auditory nerve and later decoded in the auditory cortex. Working as an intricate team with the IHCs, the OHCs generate the cochlear amplifier through an active mechanism. This active mechanism amplifies the movement of the basilar membrane and improves sensitivity to soft sounds. The OHCs are also responsible for improving frequency selectivity by refining the area of maximum displacement along the basilar membrane in response to a specific auditory input. Without healthy and properly functioning hair cells, the refining and transmitting of auditory stimuli are disrupted. Therefore individuals with NIHL have a disruption in signal transmission and consequently, experience a decrease in effective communication (Moore, 1996).

Depending on the intensity and duration of noise exposure, severity of damage to both the IHCs and OHCs is variable. Hair cell damage can be observed through a fairly structured process with the most severe injury initiating along the outermost rows of OHCs, specifically beginning at the basal end of the basilar membrane. With frequently recurring noise exposure, the damage continues to travel along the basilar membrane towards the apex. It also spreads inward toward the other rows of OHCs. Though the majority of damage does occur to the OHCs, the IHCs can also acquire injury from noise. However, through clinical observations, the IHCs appear to be more resistant to injury from noise exposure and are less likely to be affected when compared to OHCs (Harrison & Mount, 2001; Bohne & Harding, 2000).

Various insults can occur to the OHCs as a result of excessive noise exposure. Fracturing, fusing, or buckling of the stereocilia is commonly seen with NIHL (Harrison, 2001). Not only does this prevent sound-induced hair cell depolarization, it may also permanently damage the critical structures responsible for the transduction and transmission of an auditory stimulus to the vestibulocochlear nerve. These fine structures are located at the tips of the hair cells' stereocilia and are referred to as tip links (Talaska & Schacht, 2007).

The formation of reactive oxygen species (ROS) can also occur within the OHCs as a result of excessive noise exposure. Researchers speculate that the excessive formation of ROS is the most logical hypothesis for noise-induced hair cell death during and after noise exposure. However, researchers are still uncertain if

the formation of ROS causes cell damage or if they are a direct result of the damage (Henderson, Bielefeld, Harris, & Hu, 2006).

The formation of ROS is a normal metabolic process within cells. ROS are oxygen-based, signaling molecules that are only useful to cells when not in excess. When the latter occurs, the cells seek to maintain homeostasis within the body system. If a metabolic balance cannot be attained, the cells undergo oxidative stress and ultimately commence an organized cell death (Talaska & Schacht, 2007). This organized cell death can occur through two different processes. These processes are referred to as necrosis and apoptosis. Necrosis is the passive process of cell death where the cell swells and ruptures. The byproduct of the ruptured cell then causes damage to neighboring cells, initiating a similar response. Apoptosis, on the other hand, is the active process of cellular death where the injured cell is eradicated in a neat and orderly fashion to ensure that the lesion does not spread to neighboring cells. This process is utilized in development to rid the body of dying and unwanted cells. Both processes of cellular death, necrosis and apoptosis, are found within cochlear cells and regions damaged by excessive noise exposure (Henderson, Bielefeld, Harris, & Hu, 2006).

Hair cell dysfunction, damage, and death are direct results of excessive noise exposure (Talaska & Schacht, 2007). Along with the IHCs and OHCs, other regions of the cochlea can be affected by noise exposure. Like other body systems, the cochlea strives to maintain homeostasis. To do this, the cochlea must ensure it is oxygenated and has optimal blood flow throughout. In the presence of noise

exposure, cochlear blood vessels constrict and blood flow decreases. With this decrease in blood flow, different regions and features of the cochlea are deprived of oxygen. If this deprivation continues for an extended duration, cellular death occurs and the cochlea is unable to function (Henderson, Bielefeld, Harris, & Hu, 2006).

In severe cases of noise exposure, an individual is exposed to extreme and instantly damaging sound pressure levels. This exposure is referred to as an acoustic trauma. At the time of an acoustic trauma, an individual can undergo severe injuries to their auditory system that create a different type of oxidative stress, metabolic imbalance, and hair cell death. Injuries from acoustic trauma can be a perforated tympanic membrane, ossicular disarticulation, or extreme ripping of the basilar membrane (Cave, Cornish, & Chandler, 2007).

CHAPTER 4

Audiological Characteristics

As stated in the previous section, exposure to noise can result in a sensorineural hearing loss due to damage of integral components of the inner ear (Henderson, Bielefeld, Harris, & Hu, 2006). From an audiological standpoint, a hearing loss is observed when an individual's threshold falls or shifts from a range that is designated as being within normal limits. Though an individual has the potential of having permanent damage from exposure to noise, they could also exhibit a temporary hearing loss, otherwise known as a temporary threshold shift (TTS) (Quaranta, Portlatini, & Henderson, 1998).

A TTS is created when a person is exposed to low levels of damaging noise for an extended period of time. A TTS occurs due to exhaustion and metabolic stress placed on the hair cells. After a period of time isolated from excessive or damaging noise, hearing thresholds recover. If the exposure continues without the appropriate amount of rest, a permanent threshold shift (PTS) could occur (beginning with damage to the OHCs). Because of the configuration of the cochlea and the resonant frequencies of the outer and middle ear systems, the basal OHCs are affected first, specifically the OHCs that respond to 3kHz - 6kHz inputs. Because

of this, individuals with NIHL often exhibit an audiometric notch (poorer hearing thresholds) centered on 4kHz. With continued exposure to high-level noises, the hearing loss may become more severe and PTS may occur in adjacent frequencies (Quaranta, Portalatini, & Henderson, 1998).

A benchmark study by Davis, Morgan, Hawkins, Galambos, and Smith (1950) focused on exposure frequency and its effect on the hearing loss. Davis et al. created a TTS in their subjects by introducing them to excessive levels of noise at different frequencies. From this, they observed that depending on the duration of noise exposure, the greatest reduction in sensitivity occurs one half to two octaves above the exposure frequency with very little damage to cells responsible for frequencies below the exposure frequency. As the exposure continues, a broader range of frequencies becomes affected.

Besides hearing loss, another symptom of damage from excessive noise exposure is tinnitus. Tinnitus is the perception of sound without a known external source. Though tinnitus is not present in all individuals with NIHL, there is a high prevalence of it in this population of individuals, which suggests that the damage done on the structures of the inner ear from excessive noise exposure may also cause the initiation of tinnitus (Mazurek, Olze, Haupt, & Szczepek, 2010).

The final symptom of NIHL of on the auditory system is abnormal growth of loudness or loudness recruitment. Because individuals with NIHL have a sensorineural hearing loss, there is a reduction in their dynamic range. Their threshold levels are elevated, but the level where sounds become uncomfortable

does not change or lower. Individuals with recruitment report a rapid growth of loudness in response to an intense sound. Individuals who develop NIHL from an impulse noise almost always exhibit loudness recruitment due to the fact that the auditory system is unable to adapt to the instant decrease in dynamic range. Without this adaptation, individuals tend to suffer from hyperacusis and become overly sensitive to higher-level stimuli (Burke & Creston, 1966).

CHAPTER 5

Susceptibility

The risk and severity of NIHL differs among individuals due to their overall noise exposure and how their body reacts to this exposure. This reaction varies among individuals due to many biological and environmental factors including genetics, gender, pigmentation, and chronic conditions. Factors present in one's environment such as the use of tobacco products and exposure to ototoxic solvents and substances also influence the effects of NIHL (Van Eyken, Van Camp, & Van Laer, 2007).

When studying noise exposure and its effects on the auditory system, it is extremely difficult for researchers to control the noise exposure variability among individuals. Because of this known variability, most research on the overall genetic influence of the risk and development of NIHL is focused on and around research with animal models like mice, rats, and guinea pigs. When using mice for NIHL research, researchers found that some strains of mice are more susceptible to the effects of noise exposure than others, which is similar to human susceptibility. Using animals, researchers have the ability to manipulate the duration and exposure

levels of noise to pinpoint genetic influences on NIHL (Konings, Van Laer, & Van Camp, 2009).

The phenotype of every individual is created from an innumerable amount of genes responsible for his/her complete genetic make-up. Auditory researchers are focusing on genes exclusive to the cochlea and the structures responsible for receiving and encoding incoming auditory stimuli. An area of focus in this research is oxidative stress from ROS, the damage they do to cochlear structures, and how to halt this damage. Though the formation of ROS is a normal metabolic activity, too much ROS leads to irreversible damage on cells. With the help of animal research, specifically on the effects of ROS formation from noise exposure, researchers are beginning to locate and map specific genes in humans, which may provide protection against excessive ROS formation. Researchers are using this information to study different forms of antioxidants and their potential for prevention of and intervention in NIHL. With ongoing genetic mapping from animal research, researchers hope that they will one day pinpoint the exact genetic variable responsible for NIHL in humans as well as protective mechanisms (Konings, Van Laer, & Van Camp, 2009).

Individuals may be more susceptible to NIHL due to their gender and race. When correcting for age, researchers found at least a 20dB difference in PTS between males and females with the same occupational exposure (Gallo & Gorig, 1964; Berger, Royster, & Thomas, 1964). Though this research shows a gender difference between males and females, other factors like hormones and lifestyle

differences may influence susceptibility and development of NIHL. Looking at populations of men and women, Hultcrantz, Simonoska, and Stenberg (2006) deduced that women may have better hearing thresholds than men due to the female hormone estrogen. In their study, auditory brainstem response (ABR) amplitudes and latencies were measured in premenopausal and post-menopausal women. They compared the amplitudes and latencies to men of the same age and found premenopausal women had larger amplitudes and shorter latencies than men. When they assessed the women after menopause, amplitudes and latencies were similar to those of their male counterparts. To prove that the female hormones were responsible for these objective differences, Hultcrantz et al. also evaluated women with Turner's syndrome. Turner's syndrome is a condition in which women have ovarian problems resulting in low levels of estrogen. Women with this condition also have auditory dysfunction including sensorineural hearing loss. ABR testing in women with Turner's syndrome showed increased latencies closely related to the latencies obtained from the male subjects. Although these results suggest hormones are responsible for the gender differences in hearing, some women are thought to develop greater hearing loss due to use of some hormone replacement therapies and oral contraceptive pills (Hultcrantz, Simonoska, & Stenberg, 2006). Because of this contradictory information, more research is warranted to determine if female hormones protect against hearing loss and are therefore the main determinant in why women generally have better hearing than men.

Another intrinsic variable that may decrease susceptibility of NIHL is a person's race. This variable mainly stems from the color of their skin due to the concentration of melanin. Melanin is a natural substance that gives color to hair, skin, and eyes. Melanin is also located within other anatomical structures like the cochlea. The concentration of melanin in the cochlea is dependant upon the concentration in the rest of an individual's body. Melanin is thought to be a protective agent against NIHL and age-related hearing loss. The effects of melanin concentration on hearing loss have been studied in individuals with blue, green, and brown eyes as well as Caucasian and African-American individuals. Research has found that melanin in eyes has less of an effect on hearing loss than the melanin concentrations in skin (Humes, 1984). Individuals with more melanin may be more resistant to NIHL. Jerger, Jerger, Pepe, and Miller (1986) assessed the hearing thresholds of both Caucasian and African-American males with the same occupation, age, income level, education, recreational noise exposure, and duration of occupational noise exposure. They found that the African-American subjects were less susceptible to NIHL than the Caucasian subjects. Though the well-controlled study revealed a significant race difference, unknown extraneous and genetic factors and individual variability leave research without complete proof that the melanin concentration was the main determinant between the Caucasian and African-American subjects. It only suggests that melanin may provide some protection against NIHL (Jerger et al., 1986).

Not only does gender and race play a large role in the susceptibility of NIHL, but the presence of chronic conditions like hypertension, high cholesterol, and diabetes as well as the use of tobacco products may play a role in whether an individual is more susceptible to the effects of hearing loss. Agrawal, Platz, and Niparko (2009) used data obtained from the National Health and Nutrition Examination Survey (NHANES) and found that diabetes and the use of tobacco products doubled the risk of hearing loss across the entire frequency spectrum tested. This means that the entire cochlea, base to apex, is involved. Adding noise exposure, the subjects who regularly used tobacco had a common audiological configuration showing the most significant hearing loss at 8kHz and above. Subjects who did not use tobacco and had excessive noise exposure exhibited a 'noise notch' between 2kHz and 6kHz with recovery at 8kHz. This led Agrawal et al. to deduce that smoking and noise exposure may have a synergistic effect on an individual's hearing loss and could lead to damage in sections of the cochlea beyond ones typically affected in nonsmokers. Another study, Wild, Brewster, & Banerjee (2005), specifically evaluated the effects of long-term smoking on NIHL. Using a sample of male factory workers, they controlled for age, noise exposure duration, smoking habits, and medical history. They found a significant increase in thresholds of the smokers compared to the non-smoking subjects. The increases in thresholds were only apparent at 3kHz and 4kHz, while all other frequencies were similar between groups. Taking into account audiological patterns of NIHL, the authors deduced that smokers were more susceptible to hearing loss, specifically from noise. They

attributed this increase in susceptibility to smoking related damages within the cochlea to ischemia and the introduction of toxic levels of carbon monoxide. An introduction of carbon monoxide to the cochlea likely leads to hypoxia and cell death (Wild, Brewster, & Banerjee, 2005).

Agrawal, Platz, and Niparko (2009) evaluated a population of individuals with cardiovascular risk factors such as hypertension and high cholesterol. They found that individuals with cardiovascular problems had increased susceptibility for NIHL possibly due to poor circulation and insufficient oxygen supply in the cochlea. Although these effects were not as robust as those seen with tobacco use, individuals with cardiovascular risk factors had increased auditory thresholds.

Though individuals cannot alter their genetic make-up or anatomy to reduce the risk and severity of NIHL, they do possess the ability to control some environmental factors. As previously discussed, limiting the use of tobacco products not only reduces an individual's susceptibility for NIHL, but it also reduces the risk for other chronic conditions. Another environmental factor that can increase an individual's risk of NIHL is prolonged exposure to ototoxic solvents and substances. Unfortunately, it may not be possible to avoid toxic solvents and substances all together, as some individuals may be exposed to them for medical purposes or while at their occupation.

Individuals exposed to hazardous chemical solvents may exhibit other symptoms in addition to the increased risk of NIHL. Not only do these toxic chemicals affect the auditory system, but they can also create problems in the

vestibular system and central nervous system pathways. Exposure to ototoxic solvents can occur either through inhalation or skin diffusion. The auditory effect of exposure to toxic solvents is typically a bilateral sensorineural hearing loss. The combined effects of ototoxic chemical solvents and exposure to excessive noise may create a hearing loss, which is more severe in the mid-frequencies. Individuals with exposure to solvents may also exhibit increased difficulty in speech discrimination due to the solvents' effects on the central nervous system. Some of the most researched toxic chemical solvents are Toluene, Styrene, carbon disulfide, Xylene, ethyl benzene, and Trichloroethylene (Morata, Dunn, & Sieber, 1994). Because exposure to these chemicals can occur through the skin or inhalation, protection against exposure is carefully regulated. Like noise exposure, the Occupational Safety and Health Administration (OSHA) uphold strict standards and regulations to ensure at risk employees properly protect themselves against the hazardous solvents. OSHA also takes into consideration protection against noise when working with these solvents due to their interaction effects in regards to hearing loss (Hodgkinson & Prasher, 2006).

Individuals undergoing chemotherapy treatments for medical purposes are exposed to chemical agents that are ototoxic, and increase the risk for NIHL. Popular chemotherapeutic agents, cisplatin and carboplatin, are well known ototoxic substances. Depending on an individual's health and the presence of other medical conditions, both cisplatin and carboplatin can increase the risk for hearing loss. This risk tends to increase with multiple treatments, a higher dose, or a higher

concentration. Ototoxicity from chemotherapeutic agents often causes bilateral high frequency sensorineural hearing loss. This hearing loss is caused from death of auditory hair cells, damage to the stria vascularis, and a decrease in spiral ganglion cells (Li, Liu, & Frenz, 2006). With this cochlear damage and vulnerability, individuals undergoing chemotherapy are highly susceptible to further damage from excessive noise. Other medications like aminoglycosides, salicylates, and loop diuretics can also cause hearing loss. Although salicylates and loop diuretics are ototoxic, research suggests that they do not have any known interaction with noise exposure (Van Eyken, Van Camp, & Van Laer, 2007).

CHAPTER 6

Intervention & Treatment

NIHL is a permanent condition. The most effective strategy for auditory damage prevention is proper utilization of hearing protection. However, research suggests that the population of at-risk individuals has the ability to reduce the overall effects of noise exposure with the use vitamins, antioxidants, and a nutrient rich diet (Kopke et al., 2005; Le Prell, Hughes, & Miller, 2007; Le Prell Gagnon, Bennett, & Ohlemiller, 2011).

A diet full of proper vitamins and nutrients not only provides benefits against the prevention of certain medical conditions, but it can also lessen the effects of noise exposure in individuals (Le Prell, Gagnon, Bennett & Ohlemiller, 2011). With excessive noise exposure, damage to the inner ear occurs due to the formation of ROS. In excess, the ROS can result in oxidative stress leading to hair cell death (Henderson, Bielefeld, Harris, & Hu, 2006). Le Prell, Gagnon, Bennett, and Ohlemiller (2011) examined the effects of specific vitamins and their effects on the protection against damage to cells in the inner ear. They found that other than oxidative stress, cells exposed to excessive noise become damaged from the production of free radicals. Free radicals are organic molecules responsible for

tissue damage. Like ROS, free radicals are essential to many processes taking place within the body. However, damaging effects occur to cellular components with over production (Southorn & Powis, 1988). Le Prell et al. (2011) hypothesized that a diet rich in essential vitamins and antioxidants would offset the excessive formation of free radicals and therefore, result in less damage to the inner ear. After treating mice with a diet full of beta-carotene, vitamin C, vitamin E, and magnesium, the researchers found that the supplements effectively decreased inner ear cell damage responsible for a PTS after high-level noise exposure. They also concluded that combining the supplements might have synergistic effects. Other studies like one conducted by Le Prell, Hughes, and Miller in 2007 found similar results when using the same supplements in combination to reduce the damage from excessive noise.

Kopke et al. (2005) examined the effects of two specific antioxidants called N-L-acetylcysteine (NAC) and acetyl-L-carnitine (ALCAR). They administered NAC or ALCAR to adult female chinchillas pre and post noise exposure. ABR testing was performed, as well as physically counting the amount of dead hair cells. They found that the subjects treated with NAC and ALCAR exhibited smaller ABR threshold shifts accompanied by a lesser number of dead hair cells following excessive noise exposure. They also found that continuous noise exposure may be predominantly responsible for ROS formation due to the lack of hair cell death measured after exposure to impulse noise.

Currently, researchers are also looking at the effects of other medical interventions, specifically focusing on post exposure recovery treatments. One such

treatment studied by Bielefeld, Wantuck, and Henderson in 2011 assessed an Src-PTK inhibitor. Src-PTK is thought to induce initiation of apoptosis in sensory cells of the cochlea. They concluded that this drug, when taken alone or in combination with NAC, could be used as a recovery treatment post noise exposure. With further research, this drug could potentially be used mainstream to minimize the effects of excessive noise exposure.

Although interventions like antioxidants, vitamins and other supplements, and pharmacological agents are making strides with protection and rescue against excessive noise exposure, individuals that have already acquired damage to their inner ears are in need of audiologic care to address their hearing losses. Because hair cell death is irreversible and NIHL is permanent, individuals with a NIHL are optimal candidates for amplification. Due to the high frequency component observed in individuals with NIHL, communication breakdowns may occur due to increased difficulty hearing certain speech sounds. Some individuals also report a loss of interest in activities they once loved like socializing, watching television, attending church services, or listening to music. When a group of individuals with NIHL was questioned about the many things that they have difficulty hearing and how it makes them feel, Giordano et al. (2008) found that though some of their subjects felt limited and insecure in social situations, others mainly viewed their hearing difficulties as a disability rather than a handicap on their life. Though most of the subjects in this study dismissed the personal need for amplification, they

continued to view amplification as an aid for improved hearing as well as a device that can ultimately improve overall quality of life.

CHAPTER 7

Effects of Noise Exposure on Hearing in Later Life

Individuals of all ages experience adverse effects on their hearing as a result of excessive noise exposure. Children and teenagers are at a higher risk of developing NIHL due to the boom in personal music players (Vogel, Verchuure, Van der Ploeg, Brug, & Raat, 2009). The prevalence of adults with NIHL is growing due to occupational and recreational activities (Nelson, Nelson, & Conch-Barrientos, 2006; Mostafapour, Lahargoue, & Gates, 1998). However, very little is known about the effects of noise exposure on the older adult population and very little is published on NIHL in older adults. Studies like Saunders and Griest (2009) use a population of older veterans for its subjects, but most of their noise exposure was experienced many years prior to the study.

NIHL obtained in later years is difficult to evaluate due to the presence of age-related hearing loss. Diagnostic audiological data only give enough information to reveal a hearing loss. No test is present today that can separate one etiology of hearing loss from another. However, one study performed by Nondahl et al. was able to use information gathered from the Epidemiology of Hearing Loss Study performed by Cruickshanks et al. (1998). According to Nondahl et al. (2006), the

older adult population is surprisingly more active than once thought. This population continues to work until an older age. They also tend to participate in noise-rich activities like woodworking, using power tools, performing yard work, and driving noisy recreational vehicles. Because of this, older adults are at a greater risk of acquiring a NIHL than previously thought.

By assessing this population and their noisy recreational and sometimes occupational activities, Nondahl et al. discovered many things about the older adult population and their exposure to noise. They followed the study participants over a span of 10 years and ended with 2395 participants with ages ranging from 58-100 years, 41% of them being men. To collect data throughout the study, Nondahl et al. administered questionnaires regarding the participants' medical history, occupational noise exposure, recreational noise exposure, and use of hearing protection. They also performed audiometric testing to support information gathered through the questionnaires. Other than noise exposure history and use of hearing protection, Nondahl et al. also considered smoking history and history of cardiovascular disease to determine if there were any correlations between those and the likelihood of acquiring a NIHL.

After analyzing the data collected, Nondahl et al. first concluded that the prevalence of hearing loss significantly increased as the study progressed through the 5- and 10-year follow-up examinations. Next, they found that the prevalence of using hearing protection also increased throughout the course of the study. However, overall utilization of hearing protection was quite low, revealing less than

40% of the individuals used hearing protection in situations with excessive noise.

Use of hearing protection was also more accepted by individuals under the age of 65 when compared to their older counterparts. Nondahl et al. also found that females were less likely than males to wear hearing protection in noisy situations, like working in the yard or using power tools.

Damaging noise exposure can and does take place in the older adult population. NIHL will likely grow without widespread education and use of hearing protection. Because more studies focusing on this topic are scarce, further research is warranted to gain a better understanding of NIHL in older adults. Additionally, questions related to how age may or may not change susceptibility to NIHL need to be answered.

CHAPTER 8

Health Promotion

The easiest way for individuals to protect themselves against the harmful effects of noise is to completely avoid noise-rich situations (National Institute for Occupational Safety and Health (NIOSH), 1998). However, some individuals do not have the ability to make that decision due to their careers or the necessary use of certain household appliances, power tools, and/or lawn equipment (Nondahl et al., 2006). In regards to occupational noise exposure, under OSHA, specific guidelines and regulations hold strict standards and force their employees to comply by wearing hearing protection when working in excessive noise for a certain period of time (OSHA, 2008). For individuals potentially exposed to excessive leisure or recreational noise levels, the decision to wear hearing protection is now their direct responsibility (Nondahl et al., 2006).

Using information gathered from the Epidemiology of Hearing Loss Study, Nondahl et al. (2006) analyzed the use of hearing protective devices in older adults exposed to recreational noise, like hunting, woodworking/metal working, driving recreational vehicles, and using power tools. They found that overall use of hearing protective devices was low among their sample population. Although older adults

over the age of 65 generally avoid activities like hunting, they are still exposed to excessive noise from activities like woodworking and other recreational activities. From the data obtained in this study, the majority of these older adults are being exposed to dangerous noise levels without the proper use of hearing protection.

When an individual is either forced by his/her employer or decides on his/her own to wear hearing protection, there are many different styles of personal hearing protective devices available. In today's current market, there are literally hundreds of hearing protective devices to choose from. Depending on the measured level and frequency of the noise emitted, some devices may be more suitable than others. Individuals may also make the decision based on physical comfort of the device, convenience, hygiene, whether the noise is constant or intermittent, or based on activities they may be involved with during utilization of the device (Stephenson, 2009). Individuals may also want to maintain the ability to communicate effectively, hear environmental sounds while hunting, or listening to music (Peters, 2003).

Although there are numerous types of personal hearing protective devices available, the two main devices used by individuals to protect their hearing from excessive noise are earplugs and earmuffs. Earplugs range in different styles from the one-size-fits-all, disposable plugs to custom made, silicone plugs with built-in filters. All plugs are used for the same purpose, to protect one's hearing. However one style may be more beneficial than others in certain situations (Peters, 2003).

Disposable earplugs can typically be purchased from drugstores and supermarkets, or dispensed by employers, clinicians, or physicians. They are made

from a number of different materials but moldable foam or plastic is the most widely used. The earplugs fill the ear canal with an insulating material and, when inserted properly, ensure that intense sound is attenuated as it passes through the ear canal (NIOSH, 1998). Usually this type of earplug more effectively reduces high frequency noise inputs whereas earmuffs protect across the entire frequency spectrum. This configuration of protection is due to the broad shape of the low frequency waveforms and their ability to pass through the ear canal with less attenuation. Because of this, disposable earplugs are not as beneficial in conditions or environments with narrowband noise, specifically if the noise is lower in frequency (Berger & Casali, 2007).

With proper insertion, the earplugs have the ability to provide up to a maximum of 48dB attenuation (Berger & Kieper, 2003). This attenuation value is commonly referred to as the noise reduction rating (NRR). The NRR was developed by the Environmental Protection Agency and is a weighted attenuation value calculated from data obtained in laboratory conditions while using a sample population. The NRR represents attenuation values obtained by 98% of the sample population. Because these attenuation values are measured in an optimal but artificial laboratory setting, hearing protective devices seldom reach a calculated NRR in a real world environment (Berger & Kieper, 2003). Taking this information into account, both the OSHA and the NIOSH subtract anywhere from 25% to 70% of the NRR when considering possible real-world attenuation values (OSHA, 2003; NIOSH, 1998).

Real-world attenuation values can vary depending upon the insertion depth. The insertion depth of an earplug can be classified as partial, standard, or deep. Partial insertion occurs when only 20% of the plug is inserted into the ear canal, while standard insertion is when around 60% of the plug is inserted into the canal. The optimal insertion length is deep insertion, and this occurs when the earplug is 80 to 100% inserted into the canal. With a deeper insertion, greater attenuation values can ultimately be reached (Berger & Kieper, 2003).

Attenuation values can also range depending upon the specific frequency of the noise. For example, Berger and Kieper (2003) found that the mean standard deviation of deep insertion foam earplugs provide 37dB attenuation for a 2000Hz noise source and almost 48dB attenuation for a 500Hz noise source. This number could decrease considerably if the earplug is inserted either partially or at a standard depth in the ear canal.

Though disposable earplugs protect an individual's hearing in the presence of excessive noise, attenuation across a broad range of frequencies, or in the event of prolonged exposure, can only be obtained with the use of other personal hearing protection devices or double hearing protection. Double protection refers to wearing both earplugs and earmuffs simultaneously. By doing this, an individual could achieve maximum attenuation (Berger & Kieper, 2003). Attenuation values in Berger and Kieper (2003) reached over 60dB with the use of double hearing protection. Though attenuation is greater while utilizing double protection, this method of protection becomes inadequate as noise levels increase to extreme

values. At this level, exposure time would need to be significantly decreased and monitored (NIOSH, 1998).

Other than the disposable foam type, earplugs can also be customized to an individual's specific ear canal. This customization provides a tight seal within the individual's ear canal ensuring optimal attenuation from in-the-ear hearing protective devices. Reusable, custom earplugs are usually made from a soft plastic material like silicone to ensure the mold is comfortable and not abrasive within the ear canal. Custom earplugs have the ability to come in many different shapes like full shell, half shell, or canal. They can also be specifically designed for the person wearing the earplugs. Filters can be built into the molds to modify different spectral components of the noise input (Berger & Casali, 2007). This is important for individuals who would like to obtain protection while still maintaining the ability to hear communication, environmental noise, and music. Ideal users of filtered earplugs would be employees in noise-rich environments, hunters, musicians, and military personnel (Chasin & Behar, 2003).

Earmuffs are widely used hearing protective devices in both occupational and recreational environments. Earmuffs are supra-aural devices that fit securely on an individual's head. Worn correctly, the muffs completely engulf the pinna and form a tight seal. With this seal, muffs are able to be extremely effective in protecting against excessive noise across a broad range of frequencies. If worn properly, supra-aural earmuffs can potentially have an NRR of over 30dB depending on the size and style (Berger & Casali, 2007). Because earmuffs are able to provide

more attenuation over a broader range of frequencies, they should be utilized in situations where noise levels are too excessive for the use of earplugs alone.

Individuals may also choose to wear earmuffs due to comfort and ease of fit (NIOSH, 2011).

Like earplugs, there are also different styles of earmuffs. With the advances in technology throughout the years, earmuffs now have the ability to produce greater attenuation values through the use of active noise reduction (ANR). Like filters in custom earplugs, ANR circuitry in earmuffs is specifically designed to enhance the signal-to-noise ratio while maintaining adequate hearing protection. Earmuffs with built in ANR circuitry are much more effective in protection against excessive noise when compared to muffs without ANR. ANR earmuffs are quite useful to individuals who may need to communicate while wearing hearing protection (Berger & Kieper, 2003).

Hearing Conservation

According to the NHANES, it is estimated that over 18 million adults between the ages of 20 and 69 exhibit a high frequency sensorineural hearing loss, usually with a distinctive audiological notch, as a result of excessive noise exposure (Zardous, Djalilian, Rothholtz, & Bazargan, 2009). Nearly 40 million adults and

children are at risk of hearing loss due to occupational and recreational noise exposure (NIDCD, 2008).

NIHL is a completely preventable condition. The population at risk has the ability to conserve their hearing through proper preventative measures and strategies (NIDCD, 2008). However, many individuals are unaware that they are exposing themselves to harmful noise levels on a daily basis. Everyday household appliances like hairdryers, lawn equipment, and power tools are just a few culprits responsible for producing unexpectedly high noise levels. With regular and prolonged exposure to these appliances, individuals are at risk for hearing loss (ASHA, 2011).

In addition to hearing loss, tinnitus, and hypersensitivity, noise can also affect an individual's physical and emotional well-being. Jsing and Kruppa (2004) reviewed literature on the non-auditory effects of noise and found that noise not only causes annoyance, but it also increases the likelihood for an individual to develop high blood pressure or more severe cardiovascular problems, psychosocial effects, and sleep disturbances. Individuals obtaining inadequate amounts of sleep potentially face additional health and cognitive issues.

With the increasing number of individuals at risk, NIHL is evolving into an environmental risk. Millions of individuals, both children and adults, are exposed to hazardous noise levels on a daily basis (NIDCD, 2008). Without the use of hearing protection, these individuals are more susceptible to acquiring a NIHL or other detrimental effects related to noise exposure. There is a need to raise public

awareness of NIHL through the development of effective hearing health programs. These programs should not only highlight the overall effects of excessive noise exposure, but should also educate individuals about the proper methods needed to protect themselves from acquiring a hearing loss and strategies to conserve their residual hearing from further damage (Borchgrevink, 2003).

When reviewing literature on current hearing health programs implemented throughout the United States, many focus directly on the education of children and their parents about NIHL. Most of the programs are on a small scale and are generally directed toward a single school district or community (Folmer, Greist & Martin, 2002). A popular hearing health promotion program that has made huge strides throughout the years is called “Dangerous Decibels.” Dangerous Decibels was developed by the Oregon Hearing Research Center at Oregon Health and Science University, the Oregon Museum of Science and Industry, Portland State University, Portland Veteran Affairs, and the American Tinnitus Association. This program describes NIHL as a “silent epidemic,” and its main focus is to decrease the incidence and prevalence of NIHL and related symptoms in children and adolescents. When Dangerous Decibels was first introduced, it was designed to capture the attention of children through school-based, recreational, and web-based activities and provide them with information regarding different sources, consequences of, and protection from noise exposure. However, with growth and acceptance, this program now aims to provide education on a wider scale, beginning in schools nationwide and branching out into communities via audiologists and

other medical personnel, university audiology programs, as well as targeting at-risk populations in the United States like Native American communities (Martin, Sobel, Griest, Howarth, & Shi, 2006).

To assess the success of Dangerous Decibels on its target population, evaluations were performed on the different activities used to present information to children and their parents (Griest, 2008). When looking at the school-based and recreational program at the Oregon Museum of Science and Industry, the developers of Dangerous Decibels found that overall knowledge increased and attitudes and behaviors changed in both children and adults after completing the program in school or visiting the museum exhibits. They specifically found that prior to the introduction of the Dangerous Decibels program, only a small number of the children and parents wore appropriate hearing protection when needed. After participation in the program, the number of individuals wearing hearing protection, as well as the number who reported the intention to wear hearing protection, significantly increased (Martin, Sobel, Griest, Howarth, & Shi, 2006).

Since its development, the use of the Dangerous Decibels program has spread throughout the United States. Universities, school systems, and clinicians are utilizing program materials like brochures, information sheets, posters, and hands on activities to educate and ultimately lessen the effects of NIHL in children. Because this program has proven successful with children and their parents, its' goals and ideals can be used to form a health promotion program geared toward

educating the adult population about the harmful effects of dangerous noise exposure.

Unlike Dangerous Decibels, most hearing health conservation and promotion programs for adults are used in the workplace or are directed toward a specific adult population like military personnel or veterans (Pallarito, 2008). Currently, an important subpart of the United States Department of Labor, OSHA, mandates and enforces workplace health regulations and is responsible for the protection and conservation of employees' hearing health in the work environment. All employers are responsible for upholding OSHA's regulations and guidelines to their employees working in potentially dangerous noise levels and environments. These regulations and guidelines are quite specific and provide the employer and employees with a great deal of information including the harmful effects of noise and appropriate levels and exposure times. Employers must also have monitoring and audiometric screening/testing programs established. In addition to these programs, the employers must also provide hearing protective devices, training programs, and adequate recordkeeping to ensure both the employer and employees are adhering to OSHA regulations throughout the workday. Accurate recordkeeping becomes extremely important if employees develop NIHL and seek compensation. With proper education and adherence to OSHA regulations, followed by the use of appropriate hearing protective devices, consistent monitoring, and audiometric testing, it is expected that the employee should not develop a NIHL from noise exposure in his or her work environment (OSHA, 2008). However, this does become

a difficult issue when some employees still develop a significant hearing loss later in their lives. When this occurs, effects of the individual's lifestyle, susceptibility, and/or aging must also be considered.

Combining structural components of OSHA's hearing health regulations and utilizing the educational aspects of Dangerous Decibels, one could construct a successful and effective hearing health program for the prevention and conservation of NIHL in the adult population.

Prior to developing and implementing a public health program, it is important to establish a goal. In this case, the main goal is to provide adults and older adults with adequate education to ensure prevention of NIHL and hearing conservation. The target population and geographic destination can then be specified. Public health initiatives can be implemented on various population sizes. However, the population must be carefully chosen to reflect the underlying objective (Kass, 2001). For example, the Dangerous Decibels program began as a hands-on exhibit at the Oregon Museum of Science and Industry, grew to a school-based program, and is now a nationally recognized initiative where schools, institutions, universities, and clinicians use the materials to educate children and their parents in many different settings (Martin, Sobel, Griest, Howarth, & Shi, 2006). The population size should also be determined with regards to the amount of funding, while also taking into account the development, implementation, and evaluation of the program (Boeke, Zahner, Booske, & Reminton, 2008). For a vast population like all adults, young and old, around the United States, funding may

become a major issue. It has become easier for populations like veterans to receive ample education about preventative measures of NIHL since the Veteran's Administration is a federally mandated system with an allotted amount of funding for such services like prevention against NIHL (Fausti, Wilmington, Helt, Helt, and Konrad-Martin, 2005). However, in the private sector, many adults fail to consult an audiologist about hearing loss until after it has already been acquired (Kochkin, 2007). Because of this, solidifying relationships with other medical personnel, like primary care physicians would be the first step in initiating a public health initiative against NIHL and would limit funding and manpower needs. Working with primary care physicians would grant access to the adult and older adult populations. Primary care physicians must first be educated on harmful noise exposure and its detrimental effects on hearing using an evidence-based approach. Publishing NIHL studies in renowned medical journals, presenting at national meetings, and holding informational sessions may help create relationships with other medical personnel so that they can help educate our mutual patients on the harmful effects of noise exposure and proper prevention techniques and strategies. This approach has proven successful in the youth population through benchmark studies by Niskar et al. (2001) and Henderson et al. (2011) published in the Journal of the American Academy of Pediatrics.

The next step in constructing a public health program is to formulate intervention strategies necessary to reach the goal (Kass, 2001). All effective public health programs provide considerable amounts of education to its population.

Some programs may also use their funding to implement intervention or treatment, while others may only offer specific professional or social services (Nutbeam, 2000). Once the intervention strategy is determined, the program can then be implemented. In regards to a public health initiative against NIHL, education would be the main intervention strategy. Supplying adults (via their audiologist, primary care physician, or other medical provider) with a brochure or handout highlighting information on harmful levels of noise, adequate hearing protection, and NIHL would help educate this population and quite possibly reduce their incidence of NIHL.

To evaluate the effectiveness of a public health program, many factors must be considered. Not only is the structure and framework of the program assessed, but also its impact on the goal, plausibility of expected outcome, and behavioral transformation of the population (Habicht, Victoria, and Vaughan, 1999). Evaluating the effectiveness of a public health program focused on decreasing the incidence of NIHL in the adult and older adult population would prove difficult to assess due to the likelihood that the populations may have already acquired a hearing loss from noise exposure earlier in life. However, using longitudinal research on a population of adults with no known history of noise exposure, no hearing loss, and who were provided adequate education and comparing it to data obtained from a control group of subjects that lack the proper education may provide evidence of the effectiveness of the public health initiative against NIHL. The effectiveness of the program would also provide evidence as to whether or not a behavioral

transformation, ie: more frequent and consistent use of hearing protection, occurred in the target population of adults.

Due to the vast amount of people with or at risk for NIHL, the development and implementation of a hearing health program for adults should be designed on a national level. Due to scarcity of funding and manpower, assistance from other medical professionals is necessary in order to implement a program with such a large target population. The program must also be marketable, appealing, and informational to all adults ranging from young to old. A successful campaign would spark interest in adults and lead to willingness to comply with the program (Lefebvre & Flora, 1988). By utilizing all of the knowledge of successful public health programs and assessing current research on NIHL, individuals could be educated throughout their entire lives, beginning in childhood and spanning throughout adulthood.

CHAPTER 9

Conclusion

The older adult population is growing and the number of adults exposed to excessive noise levels is significant. However, with the expansive knowledge of NIHL, as well as the advancements in hearing protection and research on protective agents, there is promise that the prevalence and incidence of NIHL in the adult and older adult populations can be actively decreased. Researchers are making strides in determining what makes individuals more susceptible to NIHL. The use of different vitamins and antioxidants to lessen the susceptibility of NIHL or help rescue damaged hair cells is encouraging. However, NIHL can be completely prevented through the proper use of adequate hearing protection. It is necessary to educate adults and older adults on noise exposure and its harmful effects on the auditory system. Noises are located all around, and often, noise exposure cannot be avoided. Implementation of a hearing health and conservation program would prove useful in helping people protect themselves against harmful noise exposure now and in the future.

LIST OF REFERENCES

- Ades, H. W. & Brookhart, H. W. (1950). The central auditory pathway. *Journal of Neurophysiology*, 13, 189-205.
- Administration on Aging. (2011). *Profiles of older Americans: 2011*. U.S. Department of Health and Human Services. Retrieved September 20, 2011, from http://www.aoa.gov/aoaroot/aging_statistics/Profile/2011/docs/2011profile.pdf.
- Agrawal, Y., Platz, E., Niparko, J. K. (2009). Risk factor for hearing loss in US adults: Data from the National Health and Nutrition Examination Survey, 1999 to 2002. *Otology & Neurotology*, 30(2), 139-145.
- American Speech-Language and Hearing Association (2011). Audiology Information Series: Noise. <http://www.asha.org/uploadedFiles/AIS-Noise.pdf>.
- Berger, E. H., & Casali, J. G. (2007). Hearing Protection Devices, in *Encyclopedia of Acoustics, Volume Two* (ed. M. J. Crocker), John Wiley & Sons Inc., Hoboken, NJ, USA.
- Berger, E. H., Kieper, R. W., & Gauger, D. (2003). Hearing protection: Surpassing the limits to attenuation imposed by the bone-conduction pathways. *Journal of the Acoustical Society of America*, 114(4), 1955-2967.
- Bielefeld, E. C., Wantuck, R., & Henderson, D. (2011). Postexposure treatment with a Src-PTK inhibitor in combination with N-l-acetyl cysteine to reduce noise-induced hearing loss. *Noise & Health*, 13(53), 292-298.
- Boeke, M. C., Zahner, S. J., Booske, B. C., & Reminton, P. I. (2008). Local public health department funding: Trends over time and relationship to health outcomes. *Wisconsin Medical Journal*, 107(1), 25-32.
- Bohne, B. A., & Harding, G. W. (2000). Degeneration in the cochlea after noise damage: Primary versus secondary events. *American Journal of Otology*, 21, 505-509.
- Borchgrevink, H. M. (2003). Does health promotion work in relation to noise?. *Noise & Health*, 5(18), 25-30.

- Burke, K. S. & Creston, J. E. (1966). Recruitment in noise-induced hearing loss. *Acta Oto-Laryngologica*, 62(1-6), 351-361.
- Cave, K. M., Cornish, E. M., & Chandler, D. W. (2007). Blast injury of the ear: Clinical update from the global war on terror. *Military Medicine*, 172, 726-730.
- Chasin, M. and Behar, A. (2003). Hearing protection. *Seminars in Hearing*, 24, 345-353.
- Cruickshanks, K. J., Wiley, T. L., Tweed, T.S., Klein, R., Mare-Perkman, J. A., & Nondahl, D. M. (1998). Prevalence of hearing loss in older adults in Beaver Dam, WI: The Epidemiology of Hearing Loss study. *American Journal of Epidemiology*, 148, 879-886.
- Davis, H., Morgan, C. T., Hawkins, J. E., Galambos, R., & Smith, F. W. (1950). Temporary deafness following exposure to loud tones and noise. *Acta Oto-Laryngologica, Supplement*, 1-57.
- Fausti, S. A., Wilmington, D. J., Helt, P. V., Helt, W. J., & Konrad-Martin, D. (2005). Hearing health care: The need for improved hearing loss prevention and hearing conservation practices. *Journal of Rehabilitation Research & Development*, 42(4) Suppl., 45-62.
- Folmer, R. L., Greist, S. E., & Martin, W. H. (2002). Hearing conservation education programs for children: A review. *Journal of School Health*, 72(2), 51-57.
- Gallo, R., & Glorig, A. (1964). Permanent threshold shift changes produced by noise exposure and aging. *American Industrial Hygiene Association Journal*, 25(3), 237-245.
- Giordano, C., Garzaro, M., Nadalin, J., Pecorari, G., Boggero, R., Argebtero, P., & Albera, R. (2008). Noise-induced hearing loss and hearing aids requirement. *Acta Otorhinolaryngologica Italica*, 28, 200-205.
- Griest, S. (2008). Evaluation of a hearing-loss prevention program. *Seminars in Hearing*, 29(1), 122-136.
- Habicht, J. P., Victoria, C. G., & Vaughan, J. P. (1999). Evaluation designs for adequacy, plausibility and probability of public health programme performance and impact. *International Journal of Epidemiology*, 28, 10-18.
- Harrison, R. V. (2001). The physiology of the cochlear nerve. In: A. F. Jahn & J. Santos-Saccchi (Eds.), *Physiology of the Ear* (2nd ed.) (pp. 549-573). San Diego: Singular Publishing Group.

- Harrison, R. V., & Mount, R. J. (2001). The sensory epithelium of the normal and pathological cochlea. In: A.F. Jahn & J. Santos-Sacchi (Eds.), *Physiology of the Ear* (2nd ed.) (pp. 285-300). San Diego: Singular Publishing Group.
- Henderson, D., Bielefeld, E. C., Harris, K. C., & Hu, B. H. (2006). The role of oxidative stress in noise-induced hearing loss. *Ear & Hearing*, 27(1), 1-19.
- Henderson, E., Testa, M.A., & Hartnick, C. (2011). Prevalence of noise-induced hearing-threshold shifts and hearing loss among US youths. *Pediatrics*, 127(1), 39-46.
- Hodgkinson, L., & Prasher, D. (2006). Effects of industrial solvents on hearing and balance: A review. *Noise & Health*, 8(32), 114-133.
- Hultcrantz, M., Simonoska, R., & Stenberg, A. E. (2006). Estrogen and hearing: A summary of recent investigations. *Acta Oto-laryngologica*, 126(1), 10-14.
- Humes, L. E. (1984). Noise-induced hearing loss as influenced by other agents and by some physical characteristics of the individual. *Journal of the Acoustical Society of America*, 76(5), 1318-1329.
- Jerger, J., Jerger, S., Pepe, P., & Miller, R. (1986). Race difference in susceptibility to noise-induced hearing loss. *Otology & Neurotology*, 7(6), 399-492.
- Jsing, H., & Kruppa, B. (2004). Health effects caused by noise: Evidence in the literature from the past 25 years. *Noise & Health*, 6(22), 5-13.
- Kass, N. E. (2001). An ethics framework for public health. *American Journal of Public Health*, 91(11), 1776-1782.
- Kochkin, S. (2007). MarkeTrak VII: Obstacles to adult non-user adoption of hearing aids. *Hearing Journal*, 60(4), 24-50.
- Konings, A., Van Laer, L., & Van Camp, G. (2009). Genetic studies on noise-induced hearing loss: A review. *Ear & Hearing*, 30(2), 151-159.
- Kopke, R., Bielefeld, E. C., Liu, J., Zheng, J., Jackson, R., Henderson, D., & Coleman, J. K. M. (2005). Prevention of impulse noise-induced hearing loss with antioxidants. *Acta Oto-Laryngologica*, 125, 235-243.
- Lefebvre, R. C., & Flora, J. A. (1988). Social marketing and public health intervention. *Health Education Quarterly*, 15(3), 299-315.

- Le Prell, C. G., Gagnon, P. M., Bennett, D. C., & Ohlemiller, K. K. (2011). Nutrient-enhanced diet reduces noise-induced damage to the inner ear and hearing loss. *Translational Research*, 1-16.
- Le Prell, C. G., Hughes, L. F., & Miller, J. M. (2007). Free radical scavengers vitamin A, C, and E plus magnesium reduce noise trauma. *Free Radical Biology and Medicine*, 42(9), 1454-1463.
- Li, G., Liu, W., & Frenz, D. (2006). Cisplatin ototoxicity to the rat inner ear: A role for HMG1 and iNOS. *Neurotoxicology*, 27(1), 22-30.
- Martin, W. H., Sobel, J., Griest, S. E., Howarth, L., & Shi, Y. (2006). Noise induced hearing loss in children: Preventing the silent epidemic. *Journal of Otology*, 1(1), 11-21.
- Mazurek, B., Olze, H., Haupt, H., & Szczepek, A. J. (2010). The more the worse: The grade of noise-induced hearing loss associates with the severity of tinnitus. *International Journal of Environmental Research in Public Health*, 7, 3071-3079.
- Moore, B. C. J. (1996). Perceptual consequences of cochlear hearing loss and their implications for the design of hearing aids. *Ear and Hearing*, 17(2), 133-161.
- Moore, B. J. C. (2007). *Cochlear Hearing Loss: Physiological, Psychological, and Technical Issues*. England: John Wiley & Sons, Ltd.
- Morata, T. C., Dunn, D. E., & Sieber, W. K. (1994). Occupational Exposure to Noise and Ototoxic Organic Solvents. *Archives of Environmental Health: An International Journal*, 49(5), 359-365.
- Musiek, F. E., & Baran, J. A. (2007). *The Auditory System: Anatomy, physiology, & clinical correlates*. Boston, Massachusetts: Pearson Education, Inc.
- National Institute on Deafness and Other Communication Disorders, (2008). Noise-induced hearing loss. <http://www.nidcd.nih.gov/health/hearing/Pages/noise.aspx>.
- National Institute for Occupational Safety and Health, (2011). Noise and hearing loss prevention: Choose the hearing protection that's right for you. <http://www.cdc.gov/niosh/topics/noise/choose.html>
- National Institute for Occupational Safety and Health, (1998). Criteria for a recommended standard: Occupational noise exposure. <http://www.cdc.gov/niosh/docs/98-126/chap6.html>

Nondahl, D. M., Cruickshanks, K. J., Dalton, D. S., Klein, B. E. K., Klein, R., Tweed, T. S., & Wiley, T. L. (2006). The use of hearing protection device by older adults during recreational noise exposure. *Noise & Health, 8*(33), 147-153.

Nutbeam, D. (2000). Health literacy as a public health goal: A challenge for contemporary health education and communication strategies into the 21st century. *Health Promotion International, 15*(3), 259-267.

Occupational Safety and Health Administration, (2003). Personal protective equipment.
<http://www.osha.gov/Publications/osha3151.html>.

Occupational Safety and Health Administration, (2008). Occupational noise exposure.
<http://www.osha.gov/SLTC/noisehearingconservation/index.html>.

Pallarito, K. (2008). Hearing loss prevention goes mainstream and every practitioner has a part to play. *Hearing Journal, 61*(8), 17-22.

Peters, R. J. (2003). The role of hearing protectors in leisure noise. *Noise & Health, 5*(18), 47-55.

Quaranta, A., Portalantini, P., & Henderson, D. (1998). Temporary and permanent threshold shift: An overview. *Scandinavian Audiology, 48*, 75-86.

Stephenson, M. R. (2009). Hearing protection in the 21st century: They're not your father's earplugs anymore. *Seminars in Hearing, 30*(1), 56-64.

Saunders, G. H. & Griest, S. E. (2009). Hearing loss in veterans and the need for hearing loss prevention programs, *Noise & Health, 11*(42), 14-21.

Southorn, P. A. & Powis, G. (1988). Free radicals in medicine. I. Chemical nature and biologic reactions. *Mayo Clinic Proceedings, 63*(4), 381-389.

Talaska, A. E., & Schacht, J. (2007). Mechanisms of noise damage of the cochlea. *Audiological Medicine, 5*, 3-9.

Van Eyken, E., Van Camp, G., & Van Laer, L. (2007). The complexity of age-related hearing impairment: Contributing environmental and genetic factors. *Audiology & Neurotology, 12*(6), 345-358.

Wild, D. C., Brewster M. J., & Banerjee, A. R. (2005). Noise-induced hearing loss is exacerbated by long-term smoking. *Clinical Otolaryngology, 30*, 517-520.

World Health Organization (2010). Occupational exposure to noise: evaluation, prevention and control.

http://www.who.int/occupational_health/publications/noise.pdf.

Yueh, B. & Shekelle, P. (2007). Quality indicators for the care of hearing loss in vulnerable elders. *Journal of the American Geriatrics Society*, 55(52), 335-339

Zardouz, S., Djalilian, H., Rothholtz, V., Bazargan, M. (2009). Prevalence and risk factors for noise-induced hearing loss. *Otolaryngology-Head and Neck Surgery*, 141(3), suppl 1, 89.